Endovascular Management of Complete Vertebral Artery Dissection Presenting with Subarachnoid Haemorrhage

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Summary

Spontaneous vertebral artery (VA) dissection may involve the intradural segment of the VA and result in subarachnoid haemorrhage (SAH). These lesions are frequently associated with recurrent SAH, and have a high mortality.

Prior to the development of endovascular techniques the majority of these lesions were treated surgically. In cases where the dissection involved the posterior inferior cerebellar artery (PICA) origin surgery was associated with significant complications including recurrent SAH from retrograde VA flow into the dissected segment above the surgical clip. We describe two cases of complete VA dissection in which the entire intradural VA was sacrificed to prevent recurrent SAH. The first case tolerated planned left PICA occlusion without developing a significant neurological deficit. The second case had infarcted the right PICA territory at presentation.

Introduction

VA dissection is a frequently recognised cause of stroke, and SAH [1,2,3,4]. We present two cases of complete VA dissection presenting with SAH, one following chiropractic manipulation for neck pain. The therapeutic options, endovascular treatment, and outcome are discussed.

Case 1

A 59-year-old man with non-insulin dependent diabetes mellitus, retinopathy, and hypertension, presented with a sudden occipital headache and vomiting. There was a three week history of neck and shoulder pain for which he had received physiotherapy and chiropractic manipulation. On examination he was overweight, with a BP of 180/100 mmHg. He was fully conscious and had no focal neurological deficit (WFNS grade 1). CT confirmed SAH with a perimesencephalic-type distribution of blood. Angiography demonstrated slow flow and multifocal asymmetric long and short segment stenoses involving the entire extra and intradural left VA, with a fusiform aneurysm at the transdural segment (figures 1, 2). The left anterior inferior cerebellar artery (AICA) was noted to be a good size. The appearances were consistent with dissection of the left VA. The patient was consented for occlusion of the intradural left VA, with left PICA sacrifice.

A 4 French catheter was placed in the right VA for diagnostic angiography during the embolisation. A 6 French guide catheter was sited in the left subclavian artery and a Prowler-14 microcatheter (Cordis endovascular) was advanced over a Quicksilver-14 guidewire (Medtronic MIS) up the dissected left VA to the vertebrobasilar junction. Sixteen Tracker-10 soft coils (Target therapeutics) were detached, packing retrogradely from the vertebrobasilar junction to the extradural segment (figures 3, 4). A left PICA perfusion defect was noted on the post embolisation runs (figures 5, 6). The patient recovered consciousness immediately following the procedure, and was anticoagulated with heparin for 48 hours. There was no neurological deficit (WFNS grade 1).

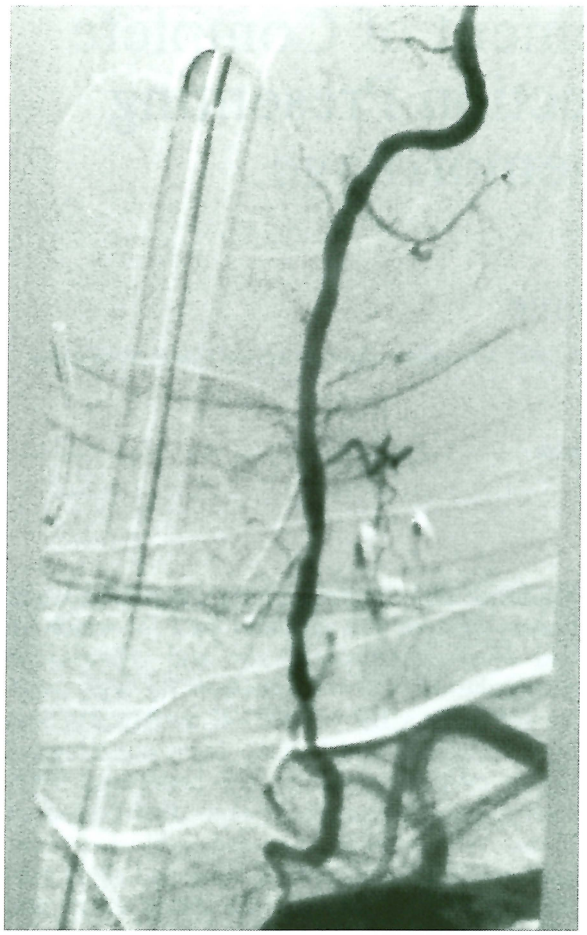

Figure 1 Left subclavian artery injection showing dissection of the entire extradural left vertebral artery.

Six weeks post procedure the patient felt well but had noticed dysaesthesia in his right arm and leg, and felt unsteady turning to the right. There was no abnormality on examination.

Case 2

A 52-year-old hypertensive man presented twenty-four hours after a sudden onset occipital headache, with unsteadiness, and a tendency to veer to the right when walking. He had stopped his antihypertensive medication of his own accord three weeks previously.

He was fully conscious with a BP of 130/80 (WFNS grade 1). CT examination was negative, but a lumbar puncture revealed blood stained CSF with xanthochromia. Angiography demonstrated slow flow and multifocal areas of luminal narrowing involving the right VA shortly after its origin, and extending to involve the entire intradural segment. A right PICA perfusion defect was noted. The appearances were consistent with dissection of the right VA, and PICA territory infarction. Using a Prowler-10 microcatheter (Cordis endovascular) and a FasDasher-14 guidewire (Target therapeutics) to access the vessel, the entire intradural segment of the right VA was sacrificed using Tracker-10 soft coils (Target therapeutics) without neurological deterioration.

The patient developed transient visual disturbance after the procedure that was attributed to migraine by an ophthalmologist. There was no neurological deterioration following the embolisation.

Discussion

Spontaneous VA dissection at any point may cause a TIA or stroke in up to 66% of cases [1,2,3]. Extracranial VA dissection may extend distally to involve the intradural segment in 20% of cases [3]. Dissection of the intradural VA segment where the media and adventitia are thinner tends to propagate in a sub-adventitial location, and frequently results in SAH [3,4]. This is particularly the case in patients with fibromuscular dysplasia [5]. Although intradural VA dissection is uncommon it is a well recognised cause of SAH, and may account for up to 3% of cases of SAH [6]. VA dissection has been identified as the cause of fatal SAH in up to 7.7% of cases in autopsy series [1].

Patients with spontaneous extracranial VA dissection frequently complain of head or neck pain [7], and may also present with stroke, Horner's syndrome, cranial neuropathy, pulsatile tinnitus, or cervical haematoma [1,8]. Recognised associations include a history of minor trauma, hypertension, anticoagulation, connective tissue disease, migraine, syphillis, and seasonal variation with a peak in the autumn months [1,5,7,9]. Spontaneous healing occurs in the majority of cases of non traumatic extracranial VA dissection, and the management advocated for such lesions varies from conservative treatment with antiplatelet drugs or formal anticoagulation, to resection of the involved VA segment [1,3,10].

Conservative management is advocated for unruptured intracranial VA dissection, but is

associated with a risk of progression to SAH [10]. In Kitinaka's series, two out of ten patients with unruptured intracranial VA dissection treated conservatively proceeded to rupture [10]. Both had risk factors for SAH, one was hypertensive, and the other was anticoagulated.

Serial angiographic follow-up was recommended for patients with non-ruptured intradural VA dissection, with progression to surgery if the angiographic abnormalities failed to resolve (persistent double lumen sign, or fusiform aneurysm), or if the patient had TIAs [10]. Anticoagulation was not recommended in these patients because of the risk of rupture and SAH.

The incidence of early recurrent SAH in patients with ruptured intradural VA dissection varies from 24 to 70% [6,11]. The mortality from a second SAH is up to 45%, and early treatment is essential in this group. The highest early rebleed rates were reported by Mitzutani [6]. Of forty-two cases of VA dissection with SAH, nineteen of the twenty nine patients treated surgically had a second SAH preoperatively. Six of the thirteen cases treated conservatively were in good condition and scheduled for surgery initially, but had a second SAH after which they were unfit for surgical management [6].

Surgical options include, proximal vessel occlusion, trapping, bleb clipping, and wrapping [11,12,13]. PICA reimplantation has been performed when trapping procedures include the PICA origin [12].

Surgical treatment may be delayed by vasospasm, and is associated with a high incidence of lower cranial neuropathy when the VA dissection extends above the PICA origin as is most commonly the case [6]. This is thought to be due to devascularisation during surgical manipulation of the cranial nerves.

Endovascular management is not precluded by vasospasm which may be treated in the same sitting, and carries a lower risk of cranial neuropathy. Anticoagulation is also a therapeutic option in patients managed endovascularly, although its benefits are not proven [1,13]. In our case 1, anticoagulation was continued after the procedure to facilitate collateral supply to the PICA territory whereas in our case 2, post procedural heparinisation was not given as the PICA had been occluded for 48 hours.

Extradural VA dissections usually originate at the C1 or C2 levels, and may extend intradurally in up to 20% of cases [3,4]. The extent of dissection in both of these cases is unusual. A similar case of extra and intradural dissection following chiropractic manipulation has been reported 13, but this did not involve the entire VA. It is not possible to prove whether chiropractic manipulation was the cause of VA dissection in case 1.

In view of our patients' medical condition, and the imaging appearances, surgical management was felt to be contraindicated. In Mitzutani's series, patients with VA dissections that involved the PICA origin had a poor out-

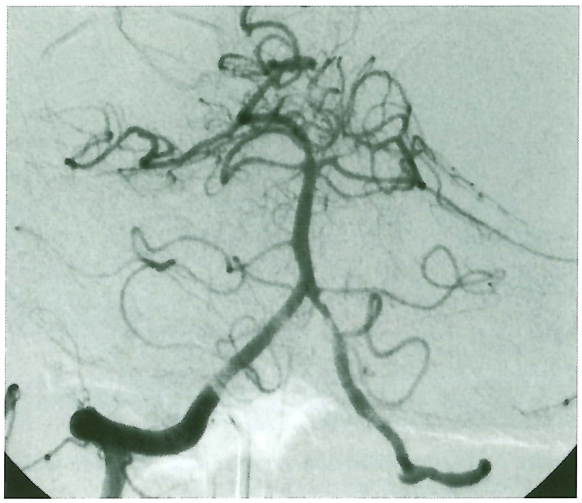

Figure 2 Dissection of the intradural segment of the left vertebral artery with a small aneurysm at the trans-dural segment.

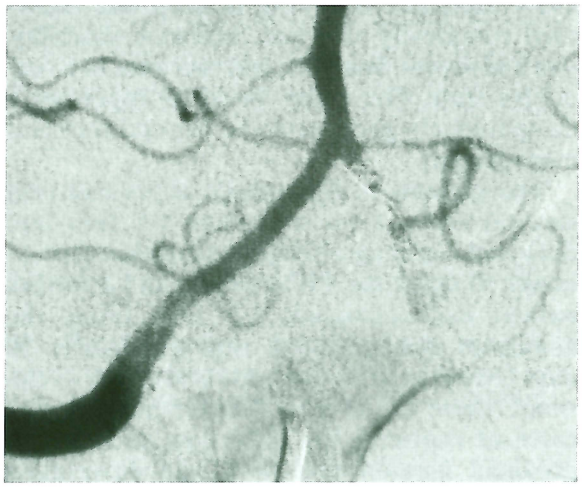

Figure 3 The first coil (Tracker-10, 2-8 soft) is positioned just short of the vertebrobasilar junction, and the origin of the left AICA.

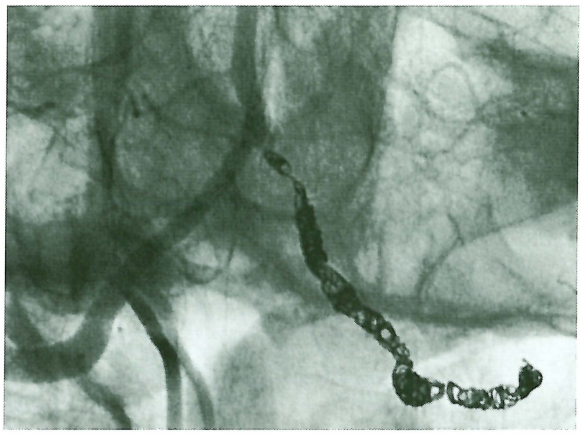

Figure 4 Post embolisation view showing occlusion of the entire intradural left vertebral artery.

come [6]. Nine had a "PICA involved" pattern of VA dissection. Three patients treated conservatively died, and following surgery, one recovered completely, two had moderate disability, one had severe disability, and two were in a vegetative state. Our cases therefore represent a comparatively good outcome for endovascular management.

Two patients in Mitzutani's series had post surgical SAHs from retrograde VA flow into dissected segments after clips were placed proximally to spare the PICA origin, and a further patient had retrograde VA flow filling the aneurysm demonstrated angiographically 50 days following proximal vertebral artery occlusion [6]. There are at least five further reported cases of ruptured VA dissection treated with proximal ligation that have had devastating post-operative SAH due to retrograde flow into the dissected segment [12,14,15,16].

The extensive VA involvement in both of our patients precluded the conventional endovascular therapeutic approach of occluding the VA at the origin of the dissection. Any therapeutic strategy had to occlude the entire intradural VA segment to avoid the risk of repeat SAH from the dissected segment via retrograde VA flow, or pial or PICA collateral supply. Parent vessel occlusion at the VA origin, and trapping the dissected segment were therefore discounted as options in these cases. In case 1, VA occlusion below the PICA origin with occlusion of the aneurysm was also discounted as it would leave the dissected segment between the PICA origin and the vertebrobasilar junction unprotected.

In view of the extent of the VA dissection, and the high risk of repeated SAH with its attendant mortality, the therapeutic aim was to occlude the entire intradural VA segment in both cases, including the left PICA origin. In case 1, the left AICA was noted to be of a good size, and it was hoped that the AICA would provide an adequate collateral supply following PICA occlusion. The absence of focal neurology despite very sluggish antegrade and retrograde VA flow demonstrated in the dissected intradural VA during the diagnostic angiogram was felt to be secondary evidence of a pial collateral circulation to the left PICA territory.

In both cases an antegrade approach to the aneurysm (accepting the risk of distal thromboembolisation) was felt to be safer than attempting a retrograde approach to the friable intradural VA segment. Tracker-10 soft coils (Target therapeutics) were selected as the least traumatic option for intradural VA occlusion, and the VA was packed retrogradely from the vertebrobasilar junction to the extradural segment without technical difficulty.

There is anecdotal evidence that accepting the risk of stroke following endovascular occlusion of the VA and PICA origin is acceptable practice in patients with VA dissection and recurrent SAH. In Halbach's series of twenty one patients with VA dissection unplanned PICA occlusion occurred in two cases [13]. In the first a balloon was inflated at the origin of a VA dissection occluding the PICA. This was associated with a mild gait disturbance, and diplopia which resolved within two months. In the second the dissection was perforated while trying to position a balloon above the PICA origin during the procedure. The PICA origin was included in the subsequent coil embolisation resulting in a lateral medullary syndrome with good recovery within two months. It seems likely that endovascular occlusion of the PICA origin in patients with VA dissection has a better outcome than surgery as pial collateral pathways are not disrupted by an endovascular approach.

PICA bypass surgery would have been an option in case 1 in an institution with a suitably skilled neurovascular surgeon. However such surgery has attendant risks, and there are no series documenting its success in this scenario. Fortunately despite PICA occlusion both patients made slow but uneventful recoveries. Both cases had symptoms of right sided dysaes-

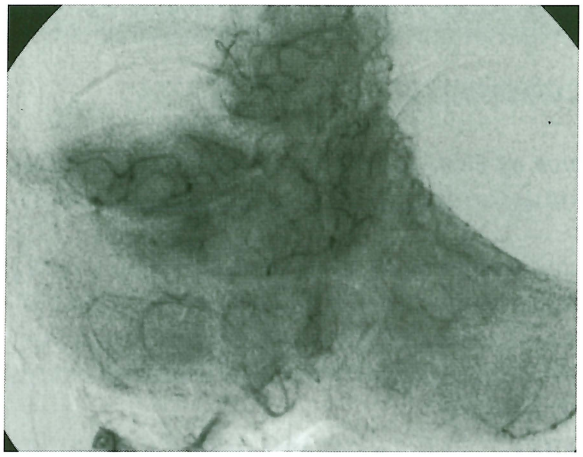

Figure 5 Pre embolisation view of the parenchogram phase of a right vertebral artery run.

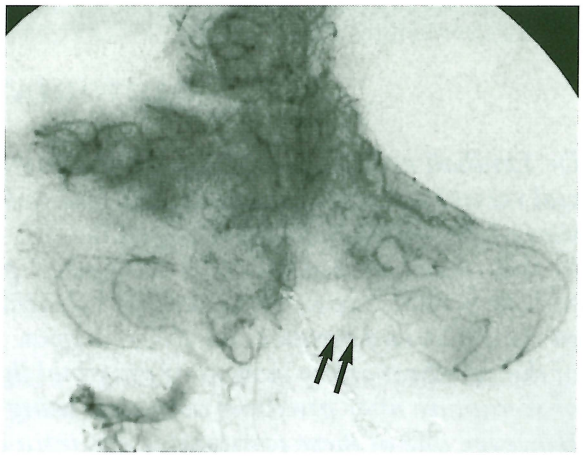

Figure 6 Post embolisation view of the parenchogram phase of a right vertebral artery run showing a left PICA perfusion defect (arrows).

thesia and imbalance suggestive of lateral medullary injury, but six weeks following embolisation both patients felt well, and were self caring. Case 1 had no deficit on examination, and case 2 had mild cerebellar signs.

Conclusion

Dissection of the intradural VA presenting with SAH is associated with early rehaemorrhage and a high mortality. Proximal occlusion of the VA sparing the PICA origin carries the risk of recurrent SAH from retrograde VA flow into the dissected segment. Complete intradural VA embolisation with PICA sacrifice is the only therapeutic option that negates the risk of potentially fatal recurrent SAH. This option, and the potential neurological sequelae should be considered in patients with VA dissections extending beyond the PICA origin.

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EDITORIAL COMMENT

Dr Lenthall et Al have highlighted the importance of early treatment of ruptured intracranial vertebral artery dissections. The report demonstrates the efficacy of endovascular occlusion of the vertebral artery using coils.

In this report an endovascular "trapping" procedure has been done in two patients using coils to occlude the entire intradural segment. In one patient the trapping procedure included the PICA and resulted in a PICA infarct. Lenthall's rational for endovascular "trapping" of the vertebral artery instead of proximal ligation is based on references to anecdotal cases of re-rupture after proximal occlusion using surgical clisp.

However, one of these re-ruptures was intra-operative[1]. In the other two cases the dissecting aneurysm was distal to PICA and the surgical clips were put either proximal or distal to PICA[2,3]. The controversy highlights the fact that management in an individual case cannot be based on generalizations from the literature.

The location and morphology of each dissecting aneurysm is unique, as is the balance between the ipsilateral PICA and AICA as well as between ipsilateral PICA and contralateral PICA. In addition, the presence or absence of a pseudoaneurysm and its location relative to PICA are important factors to consider when predicting the haemodynamic stresses that will occur after proximal occlusion. Treatment of a large vertebral artery pseudoaneurysm incorporating the PICA should be different from the treatment of an irregular stenosis in the distal vertebral artery.

Our experience is that remodelling of the distal vertebral artery occurs after proximal occlusion of a dissecting vertebral artery. Our treatment strategy includes preservation of the PICA if possible. Endovascular treatment of ruptured vertebral artery dissections should be tailored to each individual patient with the goal being the elimination of the haemodynamic stresses that prompt re-ruptured and preservation of the PICA.

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